

REMARKS

Amendment summary

Claims 23-25 are added, and are supported by, e.g., at least page 5, lines 24-28 and Example 1 of the present specification.

No new matter is added by this Amendment, and Applicant respectfully submits that entry of this Amendment is proper.

Priority Documents

Applicant respectfully requests that the Examiner indicate receipt by the USPTO of Applicant's priority documents.

Response to rejection of claims 10-11 and 22 under 35 U.S.C. § 103 based on Mae

Claims 10-11 and 22 were rejected under 35 U.S.C. § 103(a) as allegedly being unpatentable over Mae et al. (U.S. Patent No. 6,184,255) (hereinafter "Mae") in view of JP 52042837. Applicant respectfully traverses.

The present claims recite a method for protecting liver functions of a mammal, which comprises administering to a mammal the compositions recited in the claims. As discussed in the present specification, the present inventors have found that both oxidized coenzyme Q and reduced coenzyme Q have the ability to protect liver function. This can be seen by the fact that the increase in GPT or GOT activity that is normally induced by the administration of carbon tetrachloride may be suppressed by both oxidized coenzyme Q and reduced coenzyme Q. Figure 1 of the present specification illustrates this point. Figure 1 illustrates that when oxidized coenzyme Q₁₀ or reduced coenzyme Q₁₀ was administered, an increase in GPT activity, induced

by the administration of carbon tetrachloride, was suppressed. These results also show that the administration of reduced coenzyme Q has a stronger effect when compared to the administration of oxidized coenzyme Q.

Turning to the references, Mae discloses coenzyme Q₁₀. In particular, Figure 2 in Mae shows the total plasma concentration of the coenzyme Q₁₀ after oral administration of the various compositions.

However, Mae does not disclose or suggest that the coenzyme Q₁₀ in the plasma is transferred to the liver. Mae also fails to discuss how coenzyme Q₁₀ works in the liver. This is relevant because Applicant notes that the plasma concentration of coenzyme Q₁₀ does not necessarily correlate to the degree of transfer of coenzyme Q₁₀ to organs. Thus, reading Mae, a person having ordinary skill in the art would not have a reason to use either oxidized or reduced coenzyme Q₁₀ to protect liver functions, as recited in the present claims. Further, there is nothing in Mae that would lead a person having ordinary skill in the art to predict that the administration of a reduced form of coenzyme Q₁₀ provides a better efficacy in the liver when compared to the administration of an oxidized form of coenzyme Q₁₀.

JP '837 does not remedy the above deficiencies in Mae. JP '837 is cited within the Office Action for its teaching that 2,3-dimethoxy-5-decaprenyl-6-methyl-1,4-benzoquinone (oxidized coenzyme Q₁₀) is useful against hypertrophy of the liver and heart attack. This teaching does not fix the problems with Mae for the following reasons.

First, JP '837 discloses a process for preparation of oxidized coenzyme Q₁₀, but does not show any experimental data indicating the efficacy of oxidized coenzyme Q₁₀. JP '837 also fails to discuss reduced coenzyme Q₁₀. Instead, JP '837 merely teaches that oxidized coenzyme Q₁₀ may be applicable to hypertrophy of the liver.

Second, Applicant notes that hypertrophy of the liver, discussed in JP '837, is not necessarily associated with the deterioration of liver functions, and is not necessarily a disease. In this regard, Applicant notes that exaltation of metabolic function or splachnoptosia may also cause hypertrophy of the liver.

Third, JP '837 fails to disclose or suggest that something that is merely useful against hypertrophy of the liver would have the effect of protecting liver functions, as recited in the present claims. Accordingly, this element of the claims is not disclosed or taught by any of the cited references.

Finally, JP '837 teaches that an oxidized coenzyme Q₁₀ is effective for the prevention and treatment of congestive heart failure, lung congestion, and angina pectoris, in addition to hypertrophy of the liver. Applicant submits that from this teaching in JP '837, a person having ordinary skill in the art would interpret JP '837 as teaching that hypertrophy of the liver is a symptom which is associated with cardiac disorders.

Accordingly, considering the teachings of the cited references, a person having ordinary skill in the art would not have a reason to use oxidized coenzyme Q₁₀ to protect liver functions and also would not expect that oxidized coenzyme Q₁₀ would protect liver functions. A person having ordinary skill in the art would also not predict from the teachings of the cited references that the administration of a reduced coenzyme Q₁₀ has a stronger effect as compared to the administration of an oxidized coenzyme Q₁₀.

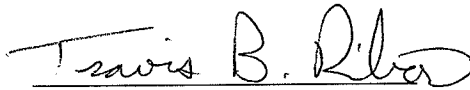
As discussed above, the presently claimed invention is not rendered obvious by the cited references, and Applicant therefore respectfully requests the reconsideration and withdrawal of this § 103 rejection.

Conclusion

In view of the above, reconsideration and allowance of this application are now believed to be in order, and such actions are hereby earnestly solicited. If any points remain in issue which the Examiner feels may be best resolved through a personal or telephone interview, the Examiner is kindly requested to contact the undersigned at the local Washington, D.C., telephone number listed below.

The USPTO is directed and authorized to charge all required fees, except for the Issue Fee and the Publication Fee, to Deposit Account No. 19-4880. Please also credit any overpayments to said Deposit Account.

Respectfully submitted,



Travis B. Ribar
Registration No. 61,446

SUGHRUE MION, PLLC
Telephone: (202) 293-7060
Facsimile: (202) 293-7860

WASHINGTON OFFICE

23373

CUSTOMER NUMBER

Date: January 15, 2009